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Search Results -

Term	Documents
CONCURRENT.USPT.	46088
CONCURRENTS.USPT.	1
TOLERAN$	0
TOLERAN.USPT.	2
TOLERANACAE.USPT.	1
TOLERANACE.USPT.	4
TOLERANACES.USPT.	2
TOLERANC.USPT.	2
TOLERANCCE.USPT.	5
TOLERANCCES.USPT.	1
TOLERANCE.USPT.	104737
. . . . .	
ANTIBOD$(ANTIBODY-WGA).USPT.	pickup term
((CONCURRENT) AND (TOLERAN$ OR TOLEROGEN$) SAME (ANTIBOD$)).USPT.	73

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Database:
- US Patents Full-Text Database
- US Pre-Grant Publication Full-Text Database
- JPO Abstracts Database
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- IBM Technical Disclosure Bulletins

Search: L6

Refine Search

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Search History

DATE: Wednesday, July 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT; PLUR=YES; OP=ADJ			
L6	(concurrent) and (toleran$ or tolerogen$) same (antibod$)	73	L6
L5	(concurrent) and (antibod$) same (antigne$ or immunogen$ or tolerogen$ or allergen) same (inhibit$ or suppress$ or block$)	179	L5
L4	(concurrent) same (antibod$) same (antigne$ or immunogen$ or tolerogen$ or allergen)	55	L4
L3	6087329.pn.	1	L3
L2	6056956.pn.	1	L2
L1	5942229.pn.	1	L1

END OF SEARCH HISTORY

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L5: Entry 121 of 179 File: USPT

DOCUMENT-IDENTIFIER: US 5762943 A
TITLE: Methods of treating type I hypersensitivity using monophosphoryl lipid A

Brief Summary Text (6):
Allergen immunotherapy in the form of a desensitization regimen is also widely used for treatment of individuals afflicted with clinically significant type I hypersensitivity reactions. Over a period of time, the individual is inoculated with small amounts of the offending allergen in an effort to achieve clinical hypo-responsiveness or desensitization against the allergen. Typically, the initial dose of the allergen is very low and gradually increases to a so-called maintenance dose which may be continued for months or years. While this type of immunotherapy has become commonplace for the treatment of sufferers from atopic allergies (including hay fever, insect sting allergy and some forms of asthma), it has significant disadvantages. Of greatest concern is the risk of a severe allergic reaction from the administration of the allergen. This inherent risk of a severe allergic reaction, such as anaphylactic shock, dictates that the treatment regimen initially employ very low amounts of allergen and only gradually increase the dose of allergen, thus prolonging the length of treatment and increasing the number of injections necessary to achieve satisfactory results since results with the treatment as now used are dose-dependent. Thus, the overall success of immunotherapy has been limited, and clinical management often focuses on control of symptoms with medications, rather than modulation of the allergy cascade by immunologic methods such as allergen immunotherapy. In addition, in patients with persistent allergic reactions, desensitization procedures are often employed with mixed results. Allergen immunotherapy elicits complex immunological responses, including the stimulation of blocking antibodies (mainly IgG) which neutralize the allergen, the alteration of the host response from a TH.sub.2 to more of a TH.sub.1 response, the stimulation of an initial rise and then gradual decrease in IgE antibodies to the specific allergen injected, the stimulation of specific anti-idiotypic antibodies, and a decrease in the allergic-type inflammatory response.

Brief Summary Text (9):
The present invention is directed to a method of treating or preventing type I IgE-dependent hypersensitivity in individuals and compositions for such treatment. The method comprises administering to the individual an effective amount of monophosphoryl lipid A (MLA) or 3-deacylated monophosphoryl lipid A (3D-MLA). It has been surprisingly discovered that MLA or 3D-MLA administered to an individual afflicted with type I hypersensitivity reduces total or allergen-specific IgE while favorably inducing the production of IgG antibodies. IgG antibodies are blocking antibodies which reduce allergic reactions. MLA or 3D-MLA may be dispensed in an effective amount in a suitable vehicle in accordance with a suitable regimen alone or administered with an allergen as part of an allergen-specific type I hypersensitivity desensitization regimen. These compounds may also be added to a vaccine composition to elicit benefits in terms of IgE-dependent allergy as well as enhance the vaccine effect. The administration of MLA or 3D-MLA results in a reduced risk of potentially serious and even fatal allergic reactions in hypersensitive individuals upon exposure to an allergen(s) to which the individual is hypersensitive.

Brief Summary Text (14):
It has been discovered that the administration of MLA or 3D-MLA in an effective amount dispensed in a suitable vehicle substantially reduces the titer of IgE class antibodies, thereby reducing the amount of IgE antibody available to attach to mast

cells which effectively reduces the release of histamine and other mediators. While not being bound to any specific theory, it is believed that the administration of MLA or 3D-MLA may stimulate the elaboration of allergen-specific IgG and reduce the elaboration of IgE antibody. The IgG would then bind and neutralize the allergen, essentially blocking the IgE-allergen interaction which initiates the hypersensitivity reaction. The reduced IgE lowers the tendency for the individual to have an allergic reaction from the allergen.

Brief Summary Text (39):
When a composition containing MLA or 3D-MLA is co-administered with an allergen(s) to treat type I hypersensitivity as part of a desensitization regimen, such composition may be administered from twenty-four hours before to twenty-four hours after administration of the allergen(s), and preferably from one hour prior, to concurrent with the administration of the allergen(s).

Detailed Description Text (16):
From the foregoing examples, it can be seen that the compositions and methods embodied by the present invention are effective to significantly reduce the level of IgE antibody associated with type I hypersensitivity in response to exposure to allergen(s) and are further effective to stimulate the production of blocking IgG antibodies, thereby reducing the risks and severity of allergic reactions upon exposure to the allergen(s) to which the patient suffers hypersensitivity.

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L5: Entry 141 of 179 File: USPT

DOCUMENT-IDENTIFIER: US 5670626 A
TITLE: Allergen-specific human IgA monoclonal antibodies for mucosal administration

Brief Summary Text (6):
Although the synthesis of allergen-specific IgE seems to be the dominant factor in the development of IgE-mediated sensitivities, the amount or the concentration of allergen-IgE is not the only factor in deciding the extent of a patient's sensitivity. Patients and persons who are exposed to but not sensitized to an allergen are known to produce IgG specific to the allergen. It is believed that these allergen-specific IgG serve as protective, blocking antibodies. People with IgA deficiency are more prone to develop IgE-mediated allergies, suggesting that IgA secreted to the mucosal surfaces of the respiratory tract can neutralize or block the trapped allergen particles or molecules, reducing their entry into the tissue. In the widely used desensitization immunotherapy treatment, patients are immunized with small amounts of allergens over a course of several months to several years. While this therapy is effective in alleviating symptoms in about haft of the allergic patients, it is not well understood by what mechanisms it works in those patients. One favorable explanation is that the treatment induces IgG blocking antibodies, because elevated levels of these antibodies can be detected in the treated patients.

Brief Summary Text (10):
The invention relates to the treatment of IgE-mediated allergic diseases, such as allergic rhinitis, allergic (extrinsic) asthma, and allergic conjunctivitis, by employing antibodies to inhibit the entry of allergenic molecules into mucosal tissues ("immune exclusion"). The invention theorizes that as the particles containing the allergenic molecules are in contact with the mucous fluids on the mucosal surface, the allergenic molecules are released from the particles and are accessible for binding by allergen-specific antibodies. The binding by antibodies inhibit the allergenic molecules to be taken up by the mucosal epithelial cells. IgA antibodies are chosen because they are more resistant to proteolytic cleavage by proteases in the mucosal fluids.

Brief Summary Text (14):
The allergen-specific IgA or IgG, or their related constructs in a physiological solution or suspension are to be applied to the mucosal tissues of patients who are allergic to the allergen. The clinical benefit is that the antibodies can bind to the allergenic protein (or other molecules to be targeted) that is released from the inhaled particles. TMs binding inhibits the allergen to be taken up by the mucosal epithelial cells and hence alleviate the development of allergic symptoms. In a preferred embodiment of the invention, the allergen-specific human monoclonal IgA antibodies are prepared in a physiological buffer for applying by dropping to the noses, eyes, or ears at the amounts of 1-2 drops per nostril, eye, or ear. The concentration of the IgA in the solution is in the range of 20 to 1000 .mu.g per ml, which is equal to 1 to 50 .mu.g of antibody per drop. The IgA may also be prepared in a solution for administration with a metered dose inhaler for reaching mucosal tissues at lower parts of the respiratory tract. The IgA concentrations are in the same range. The IgA antibody may be applied every 2 to 4 hours. It may be applied with a decongestant medication.

Brief Summary Text (22):
Allergenic molecules should be substantially accessible by the allergen-specific antibodies so that their entry to the mucosal tissue is inhibited. The allergenic

proteins are soluble in aqueous medium and should be readily soluble in the mucous fluids once they are released from the particles. The mucus contains a host of hydrolytic enzymes that should aid in digesting and loosening the contents of the particles. In any event, one can assume that the mucosal epithelium does not take up the whole pollen or mite particles and that only the released, soluble allergenic molecules are pertinent in causing the allergic reactions. The hypothesis that pollen particles or house dust particles release the crucial allergenic proteins into the mucous fluids is testable. A sample of collected house dusts can be incubated with nasal mucous secretion in vitro for varying lengths of time (1 minute to 30 minutes), the mixture is then centrifuged to pellet particles. The content of Der p I protein in the supernatant is then measured by a standard immunochemical assay for Der p I (see below).

Brief Summary Text (41):
As discussed in section A, the amounts of allergens entering the mucosal surfaces of a person in most cases are very small. On the other hand, the mucous secretions are being excreted by the mucosal tissues constantly. These suggest that the solution or suspension containing allergen-specific IgA should be applied in small quantities but at frequent intervals. It is estimated that in each individual application to the nasal linings, an eye, or an ear, amounts of 1-50 .mu.g of IgA per nostril, eye, or ear will be sufficient. Depending on the rates of secretion from the nose and from the eyes, the IgA preparation may be applied at a frequency of every two to every six hours. The frequency of application can be reduced with the concurrent use of a decongestant. Because the areas of mucosal surfaces involved in allergic asthma are much larger, more IgA will need to be applied to these surfaces.

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L5: Entry 164 of 179 File: USPT

DOCUMENT-IDENTIFIER: US 5314690 A
TITLE: Method and composition for treating IgE-mediated allergies

Brief Summary Text (8):
A few treatment schemes have been devised to reduce or eliminate an allergic response. Allergen injection therapy (allergen immunotherapy) is known to reduce the severity of allergic rhinitis. This treatment is theorized to involve the production of a different form of antibody, a protective antibody which is termed a "blocking antibody". Cooke, RA et al., Serologic Evidence of Immunity with Coexisting Sensitization in a Type of Human Allergy, Exp. Med. 62:733 (1935).

Detailed Description Paragraph Table (5):
TABLE III _____ Variations in Variation skin titers in logs from last After after SP Monkey Yrs. test prior SP* as of No. Studied to SP (logs) (logs) 3/21/91 _____ .uparw. .dwnarw. 448 14 4 4 4 4 612 13 4 5 1 1 97 5 0 3 8 5 98 5 1 0 8 8 88 5 1 2 4 3 90 5 4 1 6 7 95 5 0 0 6 5 TOTALS 52 yrs. 14 15 37 33 final studied logs.uparw. logs.dwnarw. logs.dwnarw. logs**.dwnarw. _____ *Cutaneous titers done in August-September 1990 **Total of 10 years studied Summary of changes in endpoint titers to Ascaris antigen prior to and subsequent to administration of substance P and Ascaris antigen. Columns 3 and 4 show the variations in skin titers over the years each monkey was studied (shown in column 2). These titers increased 14 logs in a total of 52 years the monkeys were studied and decreased 15 logs. Thus the cumulative variation was one log over these years prior to exposure t substance P and Ascaris by aerosol. After exposure to substance P plus Ascaris, the cutaneous titers declined in all monkeys for a total of 37 logs decrease over 10 years of total observation. Minimal changes occurred over the 7 months for a final decrease of 33 logs. The results of the foregoing examples showing a decrease in IgE mediated cutaneous reactivity in rhesus monkeys were the result of a serendipitous observation while we were studying the effects of substance P and allerge on rhesus airways. The results of decline of IgE antibody occurred after aerosol administration of substance P followed by allergen in the same experiment. We, therefore, conclude from the data that the neurokinin, substance P, and allergen down regulated the productio n of IgE antibody in the pulmonary immunologic compartment, systemically or both. The mechanism involved is not yet known with certainty. Such a change may hav resulted from direct action on B lymphocytes producing specific IgE, or b inhibiting B cell production of IgE as a result of stimulating inhibitory T cells.

CLAIMS:

2. The method of claim 1 in which said concurrent administrations are periodically repeated to further reduce the mammal's allergic reaction.

Search History

DATE: Wednesday, July 24, 2002 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=JPAB,EPAB,DWPI; PLUR=YES; OP=ADJ			
L4	(gp39 or cd40L or cd40 adj ligand or 5c8) same (antibod$) and (inhibit$ or suppress$ or block$ or prevent$) same (humoral or antibod$ or immunoglobulin$) same (antigen$ or tolerogen$ or immunogen$)	24	L4
DB=USPT,PGPB; PLUR=YES; OP=ADJ			
L3	(gp39 or cd40L or cd40 adj ligand or 5c8) same (antibod$) and (inhibit$ or suppress$ or block$ or prevent$) same (humoral or antibod$ or immunoglobulin$) same (antigen$ or tolerogen$ or immunogen$)	221	L3
L2	(gp39 or cd40L or cd40 adj ligand or 5c8) same (antibod$) and (inhibit$ or suppress$ or block$ or prevent$) same (humoral or antibod$ or immunoglobulin$)	294	L2
L1	noelle-randolph$	16	L1

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Search Results - Record(s) 11 through 16 of 16 returned.

☑ 11. Document ID: US 5902585 A

L1: Entry 11 of 16 File: USPT

US-PAT-NO: 5902585
DOCUMENT-IDENTIFIER: US 5902585 A

TITLE: Methods of inducing T cell unresponsiveness to donor tissue or organ in a recipient with GP39 antagonists

DATE-ISSUED: May 11, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Durie; Fiona H.	Seattle	WA		
Parker; David C.	Grafton	MA		
Appel; Michael C.	Grafton	MA		
Phillips; Nancy E.	Shrewsbury	MA		
Mordes; John P.	Newton	MA		
Grenier; Dale L.	Hubbardston	MA		
Rossini; Aldo A.	Sudbury	MA		

US-CL-CURRENT: 424/144.1; 424/130.1, 424/133.1, 424/134.1, 424/141.1, 424/143.1, 424/154.1, 424/173.1, 514/2, 514/8, 514/885, 530/350, 530/387.1, 530/387.3, 530/388.1, 530/388.2, 530/388.22, 530/388.7, 530/388.73, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

☑ 12. Document ID: US 5876718 A

L1: Entry 12 of 16 File: USPT

US-PAT-NO: 5876718
DOCUMENT-IDENTIFIER: US 5876718 A

TITLE: Methods of inducing T cell non-responsiveness to transplanted tissues and of treating graft-versus-host-disease with anti-gp39 antibodies

DATE-ISSUED: March 2, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Foy; Teresa M.	Seattle	WA		
Aruffo; Alejandro	Edmonds	WA		
Ledbetter; Jeffrey A.	Seattle	WA		

US-CL-CURRENT: 424/154.1; 424/130.1, 424/133.1, 424/134.1, 424/141.1, 424/143.1, 424/193.1, 435/326, 435/332, 435/334, 435/343, 435/343.1, 530/388.2, 530/388.22, 530/388.7, 530/388.73, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Image

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☑ 13. Document ID: US 5869049 A

L1: Entry 13 of 16 File: USPT

US-PAT-NO: 5869049
DOCUMENT-IDENTIFIER: US 5869049 A

TITLE: Methods of inducing T cell unresponsiveness to bone marrow with gp39 antagonists

DATE-ISSUED: February 9, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Foy; Teresa M.	Federal Way	WA		
Durie; Fiona H.	Seattle	WA		
Parker; David C.	Grafton	MA		
Greiner; Dale L.	Hubbardston	MA		
Rossini; Aldo A.	Sudbury	MA		
Mordes; John P.	Newton	MA		

US-CL-CURRENT: 424/154.1; 424/130.1, 424/134.1, 424/143.1, 424/144.1, 424/173.1, 424/233.1, 514/12, 514/2, 514/8

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☑ 14. Document ID: US 5833987 A

L1: Entry 14 of 16 File: USPT

US-PAT-NO: 5833987
DOCUMENT-IDENTIFIER: US 5833987 A

TITLE: Treatment of T cell mediated autoimmune disorders

DATE-ISSUED: November 10, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Claassen; Eric	Pijnacker			NL

US-CL-CURRENT: 424/154.1; 424/130.1, 424/133.1, 424/141.1, 424/142.1, 424/143.1, 424/144.1, 424/153.1, 424/173.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

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☐ 15. Document ID: US 5747037 A

L1: Entry 15 of 16 File: USPT

US-PAT-NO: 5747037
DOCUMENT-IDENTIFIER: US 5747037 A

TITLE: Anti-GP39 antibodies

DATE-ISSUED: May 5, 1998

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Foy; Teresa M.	Seattle	WA		
Aruffo; Alejandro	Edmonds	WA		
Ledbetter; Jeffrey A.	Seattle	WA		

US-CL-CURRENT: 424/154.1; 424/130.1, 424/141.1, 424/143.1, 424/144.1, 424/153.1, 424/173.1, 435/326, 435/332, 435/334, 435/343, 435/343.1, 435/343.2, 435/346, 435/70.21, 530/387.1, 530/388.1, 530/388.2, 530/388.22, 530/388.7 , 530/388.73, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☑ 16. Document ID: US 5683693 A

L1: Entry 16 of 16 File: USPT

US-PAT-NO: 5683693
DOCUMENT-IDENTIFIER: US 5683693 A

TITLE: Method for inducing T cell unresponsiveness to a tissue or organ graft with anti-CD40 ligand antibody or soluble CD40

DATE-ISSUED: November 4, 1997

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Durie; Fiona H.	Seattle	WA		
Parker; David C.	Grafton	MA		
Appel; Michael C.	Grafton	MA		
Phillips; Nancy E.	Shrewsbury	MA		
Mordes; John P.	Newton	MA		
Grenier; Dale L.	Hubbardston	MA		
Rossini; Aldo A.	Sudbury	MA		

US-CL-CURRENT: 424/144.1; 424/130.1, 424/133.1, 424/134.1, 424/141.1, 424/143.1, 424/154.1, 424/173.1, 514/2, 514/8, 514/885

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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Term	Documents
NOELLE-RANDOLPH$	0
NOELLE-RANDOLPH.USPT,PGPB.	1
NOELLE-RANDOLPH-J.USPT,PGPB.	15
NOELLE-RANDOLPH$.USPT,PGPB.	16
(NOELLE-RANDOLPH$).USPT,PGPB.	16

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Search Results - Record(s) 1 through 10 of 16 returned.

☐ 1. Document ID: US 20020058037 A1

L1: Entry 1 of 16 File: PGPB May 16, 2002

PGPUB-DOCUMENT-NUMBER: 20020058037
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020058037 A1

TITLE: Use of anti-gp-39 antibodies for treatment and/or reversal of lupus and lupus associated kidney disease

PUBLICATION-DATE: May 16, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Noelle, Randolph J.	Cornish	NH	US	
Burns, Christopher M.	Lyme	NH	US	

US-CL-CURRENT: 424/146.1; 424/85.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw. Desc | Image

☐ 2. Document ID: US 20020048579 A1

L1: Entry 2 of 16 File: PGPB Apr 25, 2002

PGPUB-DOCUMENT-NUMBER: 20020048579
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020048579 A1

TITLE: Ex vivo treatment of allogeneic and xenogeneic donor T-cells containing compositions (bone marrow) using gp39 antagonists and use thereof

PUBLICATION-DATE: April 25, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Noelle, Randolph J.	Cornish	NH	US	
Blazar, Bruce R.	Golden Valley	MN	US	
Vallera, Daniel A.	St. Louis	MN	US	
Taylor, Patricia A.	St. Paul	MN	US	

US-CL-CURRENT: 424/144.1; 424/93.7, 435/372

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw. Desc | Image

☐ 3. Document ID: US 20020022020 A1

L1: Entry 3 of 16 File: PGPB Feb 21, 2002

PGPUB-DOCUMENT-NUMBER: 20020022020
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020022020 A1

TITLE: Ex vivo treatment of allogeneic and xenogeneic donor T-cells containing compositions (bone marrow) using gp39 antagonists and use thereof

PUBLICATION-DATE: February 21, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Noelle, Randolph J.	Cornish	NH	US	
Blazar, Bruce R.	Golden Valley	MN	US	
Vallera, Daniel A.	St. Louis	MN	US	
Taylor, Patricia A.	St. Paul	MN	US	

US-CL-CURRENT: 424/93.71; 435/372

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 4. Document ID: US 20020009450 A1

L1: Entry 4 of 16 File: PGPB Jan 24, 2002

PGPUB-DOCUMENT-NUMBER: 20020009450
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020009450 A1

TITLE: Treatment of T cell mediated autoimmune disorders

PUBLICATION-DATE: January 24, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Noelle, Randolph J.	Cornish	NH	US	
Claassen, Eric	Pijncker		NL	

US-CL-CURRENT: 424/154.1; 424/142.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 5. Document ID: US 20010033840 A1

L1: Entry 5 of 16 File: PGPB Oct 25, 2001

PGPUB-DOCUMENT-NUMBER: 20010033840
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20010033840 A1

TITLE: Methods for inducing T cell tolerance to a tissue or organ graft

PUBLICATION-DATE: October 25, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Noelle, Randolph J.	Cornish	NH	US	
Durie, Fiona H.	Seattle	WA	US	

US-CL-CURRENT: 424/144.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 6. Document ID: US 6376459 B1

L1: Entry 6 of 16 File: USPT

US-PAT-NO: 6376459
DOCUMENT-IDENTIFIER: US 6376459 B1

TITLE: Inhibiting B cell activation with soluble CD40 or fusion proteins thereof

DATE-ISSUED: April 23, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Aruffo; Alejandro A.	Edmonds	WA		
Ledbetter; Jeffrey A.	Seattle	WA		
Stamenkovic; Ivan	Brookline	MA		
Noelle; Randolph	Plainfield	NH		

US-CL-CURRENT: 514/2; 424/133.1, 514/12, 514/885, 530/350, 530/866, 530/868

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☑ 7. Document ID: US 6375950 B1

L1: Entry 7 of 16 File: USPT

US-PAT-NO: 6375950
DOCUMENT-IDENTIFIER: US 6375950 B1

TITLE: Methods for inducing T cell unresponsiveness to donor tissue or organ in a recipient with gp39 antagonists

DATE-ISSUED: April 23, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Durie; Fiona H.	Seattle	WA		

US-CL-CURRENT: 424/154.1; 424/130.1, 424/133.1, 424/134.1, 424/141.1, 424/143.1, 424/144.1, 424/153.1, 424/173.1, 424/184.1, 424/192.1, 424/577, 424/93.7, 424/93.71, 514/2, 514/8, 514/885, 530/350, 530/387.1, 530/387.3 , 530/388.1, 530/388.2, 530/388.22, 530/388.7, 530/388.73, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 8. Document ID: US 6328964 B1

L1: Entry 8 of 16 File: USPT

US-PAT-NO: 6328964
DOCUMENT-IDENTIFIER: US 6328964 B1

TITLE: Method to treat multiple sclerosis with GP39-specific antibodies

DATE-ISSUED: December 11, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Claassen; Eric	Punacker			NL

US-CL-CURRENT: 424/154.1; 424/130.1, 424/133.1, 424/141.1, 424/142.1, 424/143.1, 424/144.1, 424/153.1, 424/173.1, 530/387.1, 530/387.3, 530/388.1, 530/388.2, 530/388.22, 530/388.7, 530/388.73, 530/388.75

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments KWIC | Draw Desc | Image

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☐ 9. Document ID: US 6312692 B1

L1: Entry 9 of 16 File: USPT

US-PAT-NO: 6312692
DOCUMENT-IDENTIFIER: US 6312692 B1

TITLE: Method of treating graft-versus-host disease with anti-GP39 antibodies and bone marrow cells

DATE-ISSUED: November 6, 2001

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
Noelle; Randolph J.	Cornish	NH		
Foy; Teresa M.	Seattle	WA		
Aruffo; Alejandro	Edmonds	WA		
Ledbetter; Jeffrey A.	Seattle	WA		

US-CL-CURRENT: 424/154.1; 424/130.1, 424/141.1, 424/143.1, 424/144.1, 424/153.1, 424/173.1, 424/520, 424/577, 424/93.7, 424/93.71, 435/332, 435/334, 435/343, 435/343.1, 435/343.2, 435/346, 530/387.1, 530/388.1, 530/388.2, 530/388.22, 530/388.7, 530/388.73, 530/388.75

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TITLE: Method for prolonged suppression of humoral immune response to a

thymus-dependent antigen therapeutic agent

DATE-ISSUED: August 24, 1999

INVENTOR-INFORMATION:

NAME	CITY	STATE	ZIP CODE	COUNTRY
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